Sleep Recovery from Physical Exercise: A New Understanding of Brain Responses to Stress

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Abstract

The effects of physical exercise on human sleep (exercise in temperate conditions, in the cold and in hot climates) are analysed and discussed in comparison to studies on sedentary sleep in extreme environments (tropical and polar climates), and on sleep in rats after stressful events (sleep deprivation). An attempt to interprete the stress-induced sleep changes is developed, involving a "central" response and a "general" stress response. These responses ("diachronic" or "synchronic") are also examined in relation to chronobiological mechanisms.

INTRODUCTION

Most studies on physiological recovery after physical exercise concern immediate reactions. The number of studies dealing with long-term recovery, especially with the restorative functions of sleep after exercise is indeed limited. The effects of physical exercise on sleep patterns are still subject to controversy (4). The association of the secretory peak of growth hormone with sleep onset (39, 48) initiated the restorative theory of sleep which implied that physical exercise would induce an increased need for slow-wave sleep (38). However, although Horne (19, 20, 21, 22) agrees with the coexistence of growth hormone and the increase mitotic activity during nocturnal sleep, he denies the role of physical restoration to slow-wave sleep. Horne and Staff (23) attributed the changes in slow-wave sleep to a modification in the cerebral function. Furthermore, the after-effects of exercise on sleep depend on the fitness level of the subject, the exercise programme, and the degree of strain imposed on individuals. Sleep changes were attributed to stress-induced sleep disturbances or a "hyperthermia"-induced increase in slow-wave sleep (4). When the strain due to exercise provokes a diurnal stress reaction, marked for example by increased adrenal cortical activity, slow-wave sleep may be impaired. If the stress reaction extends through to the following night, rapid-eyemovement (REM) sleep will also diminish (8, 9). The hyperthermia effect is characterized by increased total sleep time and slow-wave sleep (23). The latter can be counteracted by body cooling during the exercise (24). This may explain why marathon runners show either no variation in slow-wave sleep after exercising in a cold climate (49) or increased amounts of slow-wave sleep, when running in a warm climate (44, 45). However, recent development in studies on sleep deprivation in animals tend to relate the rebound in sleep during recovery to a stress response modulated in the central nervous system.

Our purpose was to further examine the implication of this hypothesis on sleep changes after exercise, through experiments conducted in our laboratory on sleep in extreme environments.

METHODS

Although sleep can be approached through interviews, questionnaires and sleep diaries, the only objective method to record sleep patterns remains the polysomnographic technique. Polysomnography requires carefully placed electroencephalographic (EEG) electrodes, following the 10-20 electrode system. Other electrodes are fixed near the eye sockets to record electrooculogram, and at the tip of the chin to record electromyography, as the EEG and these two parameters are necessary for the scoring of the states of vigilance. Polygraphy may also include the recording of heart rate by electrocardiography, respiration to

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detect sleep apneas, body temperature, or even the pH of the lower cesophagus to examine gastro-cesophageal reflux. The subject can be recorded directly through a connecting cable attached to an EEG machine, or an ambulatory system such as the portable Oxford Medilog 9000 series system, which is able to record 8 channels of electrobiological signals for 24 hours on a C-120 audio cassette. The traces are scored using the international classification (42) in order to produce a hypnogram, which represents the distribution of the states of vigilance throughout the night. The states of vigilance are represented by wakefulness, REM sleep and non-REM sleep. The latter is constituted of 4 stages: stage 1 being a transitory stage, stage 2 being characterized by the occurrence of sleep spindles and K complexes, and stages 3 and 4 containing an increasing proportion of slow-waves of high amplitude (delta waves, 0.5 to 4 Hz) and are thus called slow-wave sleep. The night of sleep is rhythmic, due to the succession of 3 to 6 REM sleep episodes, equally spaced at 90 min intervals, determining 3 to 6 REM-non-REM sleep cycles. The first sleep cycle starts with sleep onset till the end of the first REM sleep episode; the second extends from the end of the first to the end of the second REM sleep episode, and so on. Under temperate conditions, in young healthy sedentary subjects, slow-wave sleep occurs during the first half of the night and mostly during the first two sleep cycles (Fig. 1). Contrarily, REM sleep episodes are longer during the second half of the night. Calculations regarding each stage of vigilance are made versus sleep period time (from falling asleep to the last awakening) for wakefulness and versus total sleep time (sleep period time minus intercurrent wakefulness) for the stages of sleep (10).

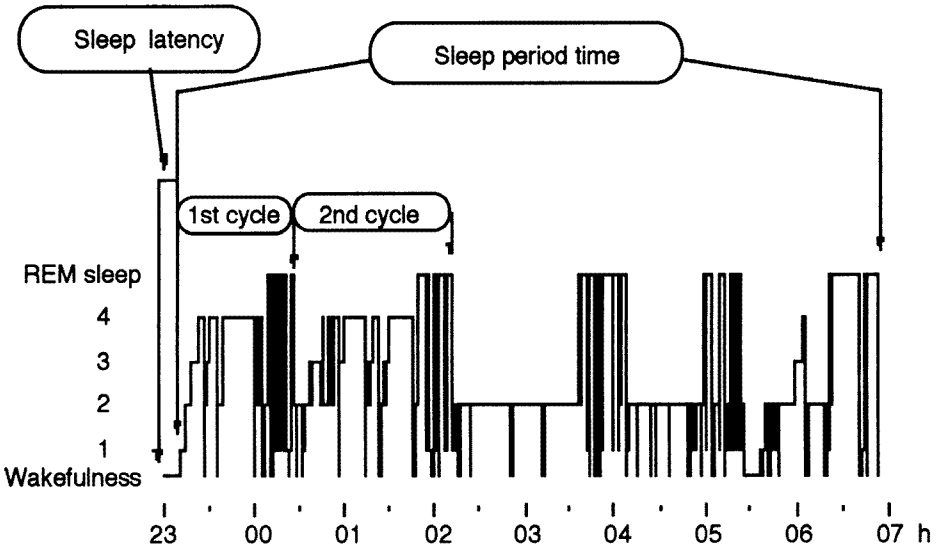

Fig. 1. Hypnogram showing the distribution of the stages of vigilance (wakefulness; REM sleep; non-REM sleep, stages 1 to 4) throughout night sleep in a young healthy subject living in a temperate climate.

In all our experiments, polysomnographic recordings were taken for 3 consecutive nights, the first night being used for subject adaptation to avoid the well known first night effect sleep disturbances attributed to the laboratory environment (1).

Under temperate conditions and in young healthy subjects, the proportions of the stages of vigilance are approximately as follows: 1-5 % of wakefulness, 1-5 % of stage 1, 45-55 % of stage 2, 16-23 % of slow-wave sleep and 18-23 % of REM sleep.

INDIVIDUAL SLEEP PATTERNS AFTER EXERCISE IN A TEMPRATE CLIMATE

Sleep patterns were studied after moderate exercise in temperate conditions under a joint experiment with the Franco-Canadian Accord for Defence Research (8). The experiment took place in the fall near Lyon. The subjects marched for 6 hours at 6 km.h-l during 6 consecutive days, between 09:00 h and 17:00 h. Each subject carried a back pack to adjust energy expenditure to 40 % of individual tO2maxX The experiment was

divided into three epochs, with a 5-day baseline, 6 days of exercise and 5 days of recovery. Sleep was recorded every night. Urines were collected from 09:00 h to 17:00 h, from 17:00 h to Z:00 h and during the night from 22:00 h to 06:00 h, and 17-hydroxycorticosteroids (17-OHCS) were analysed to serve as an indicator of adrenocortical activity.

Fig. 2 demonstrates that individuals experienced differences in sleep patterns after exercise, in relation to adrenal cortical activity. Subject S5 was a farmer for whom the march did not represent a s train, as s how n by a decrease of adrenocortical activity. He showed an increase in both stages 3 and 4 after exercise. The next 3 subjects showed an increase in stage 3 without any modification in 17-OHCS excretion during the march. Subject S4 had an increased excretion of urinary 17-OHCS, and also showed no change in stage 3. On the contrary, subject S6, who exhibited the largest increase in 17-OHCS excretion during the march, had a decrease in stage 3 and stage 4. The increased adrenocortical activity extended into the night: he was the only subject with a decrease in REM sleep.

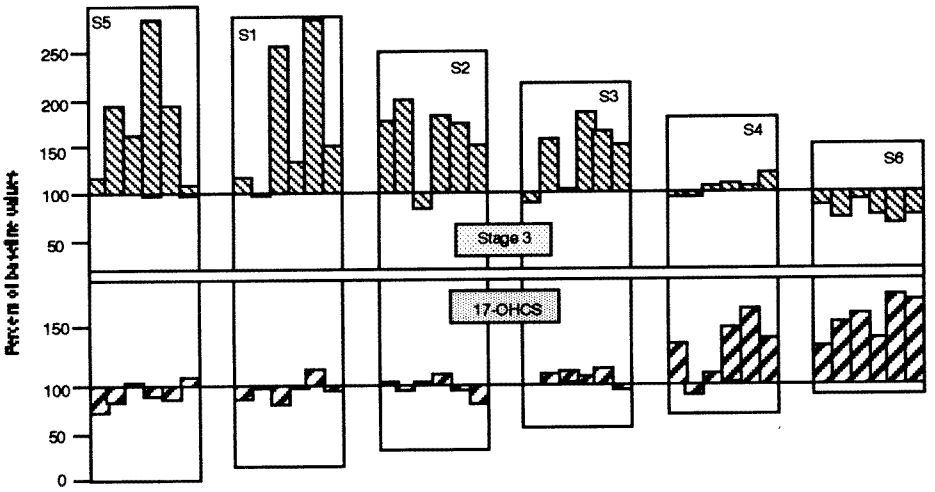

Fig. 2. Sleep pattern changes (Stage 3) in the 6 subjects (S 1 -S6) during the 6-day exercise period in relation to the daytime urinary excetion of 17 hydroxycorticosteroids (17-OHCS) while marching. The values obtained during the 6-day exercise period were expressed as a percentage of baseline values for each individual. The subjects were ranked in decreasing order of slow-wave sleep increase, which corresponds to the increasing order of 17-OHCS secretion.

These results can be interpreted in terms of the presence or the absence of a "classical" stress reaction, as evidenced by an increased heart rate during night sleep (43).

These results can also be interpreted in terms of chronobiology. In S4 and S6, two types of effects were observed. There was a decrease in total sleep time and in slow-wave sleep which was distant from exercise. This type of delayed reaction has been called "diachronic" by Jouvet (28). The second effect was the decrease in REM sleep in S6, which was concomitant with an increase in adrenocortical activity at night, i.e. a "synchronic" reaction. In the other 4 subjects, the absence of any stress reaction during the exercise led to a diachronic increase in slow-wave sleep.

This diachronic increase in slow-wave sleep could also be refered to as an effect of the exercise-induced hypertherrnia, not counterbalanced by the stress effect. It has been reported that sauna exposure (41) or a hot bath (32) induce an increase in slow-wave sleep the following night.

To answer these questions, sleep was studied in sedentary subjects and in athletes with and without physical exercise in a dry tropical climate in Africa.

SLEEP IN SEDENTARY SUBJECTS IN A DRY TROPICAL CLIMATE

The study took place in Niger over a period of 12 years, in a sahelian climate, during the dry season, because of power failure in the rainy season. In the Sahel, the dry season is divided in a cool season in January and February and a hot or very hot season in April, May and June. During the month of March, the ambient temperature increases steadily. The experiments took place at the Faculty of Medicine of Niamey. The polysomnographic recordings were realised in an airconditioned laboratory, at ambient temperatures of 23-24 °C. Sleep patterns were analysed in 34 African students in sedentary conditions and in 6 French military expatriates. All subjects were recorded by direct polysomnography during 3 consecutive nights in each of the cool and hot seasons.

The hypnograms of African subjects (Fig. 3), as well as those of European expatriates (unshown data, 35), showed that sleep architecture differed from that of people living in temperature countries(l2, 16).

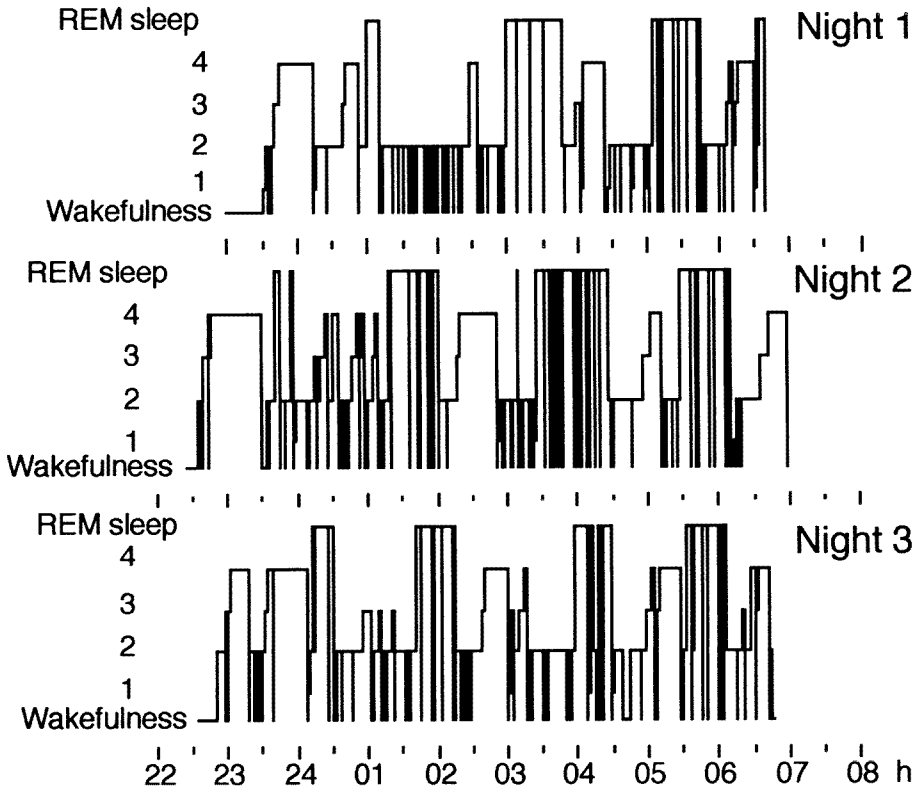

Fig. 3. Hypnograms showing the distribution of night sleep in an African subject during three consecutive nights. The states of vigilance are represented by wakefulness, REM sleep, and non-REM sleep (stages 1, 2, 3 and 4).

The modifications in the amount of slow-wave sleep were related to the ambient temperature and slow-wave sleep increased in the hot season compared to the cool season. The increase in slow-wave sleep during the hot season occurred at the expense of stage 2. This was well demonstrated in the European expatriates who were also studied during the transitory March period (Fig. 4).

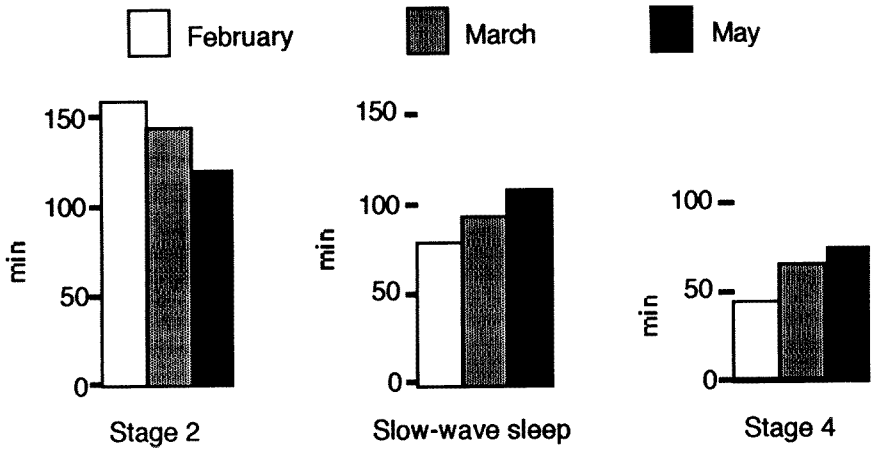

Fig. 4. The effect of the seasonal heat on sleep patterns of European expatriates during the cool (February) and hot (May) seasons and during the intermediary month of March.

In conclusion, daytime exposure to climatic heat led to diachronic changes in sleep patterns, especially an increased amount of slow - wave sleep proportional to the environmental heat. To determine whether heat-induced sleep pattern changes would be modified by endogenous heat production, the effect of exercise under such climatic conditions was analysed.

SLEEP AFTER EXERCISE IN SUBJECTS LIVING IN A DRY TROPICAL CLIMATE

The effects of progressive training were analysed in sedentary subjects (13), using a square wave endurance exercise test performed on an ergometer. The physical fitness level of the subjects (maximal aerobic power, MAP) was evaluated from a triangular maximal test with 25 W increments every 2 min performed before training. VO_{2max} was estimated from MAP values and confirmed the sedentary quality of the subjects (between 2.1 and 2.8 $L.min^{-1}$)

The training programme consisted of a 6-week square-wave endurance exercise test (SWEET) performed 3 times a week. The test was made of a repeated 5-min sequence with a 4min submaximal plateau followed by a 1-min maximal peak. The number of sequences was increased from 5 in the first week to 7 in the second week, then to 9 during the following 4 weeks. The improvement of physical performance was judged using triangular maximal tests after the third and sixth weeks of training. The third week triangular test served to adjust the work load to the improved level of MAP. All the exercise tests were performed at laboratory temperatures of 24-25 °C. The training programme was realised twice, during the cool season and the hot dry season of the sahelian dry tropical climate, with an interval of 10 weeks during which routine sedentary activity was resumed. Physical fitness improved during each training session, but had returned to baseline values after the 10-week training interruption. Baseline polysomnographic recordings were taken during 3 consecutive nights, preceding the training session. During the fourth and sixth weeks of each exercise period, sleep recordings were performed during the last 2 consecutive nights following day exercise.

After exercise (Fig. 5), slow-wave sleep increased during the cool season, compared to baseline values. This increase was enhanced during the hot season. Therefore, the conjonction of muscular heat production and external climatic heat load led to a diachronic increase in slow-wave sleep. REM sleep was not influenced by exercise nor by the season.

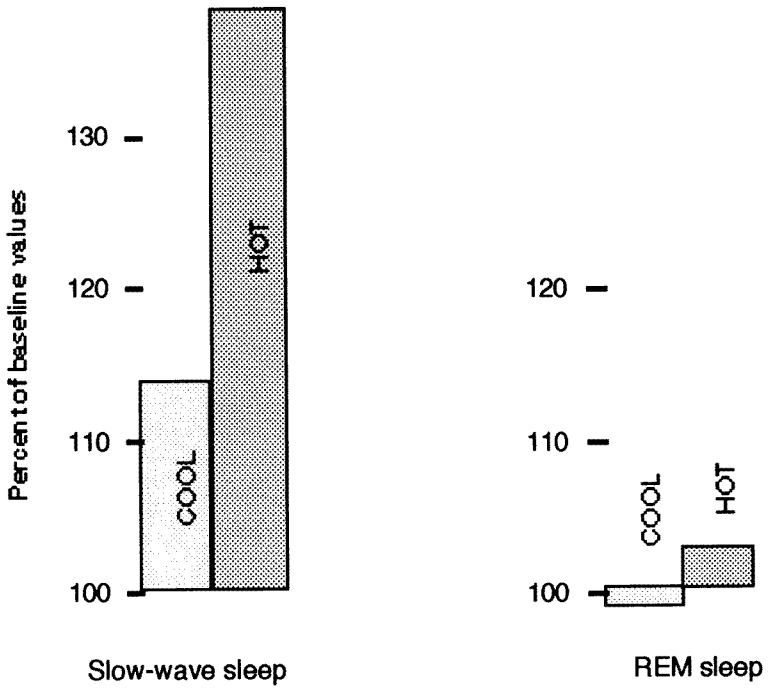

Fig. 5. Combined effect of physical training and seasonal heat variations on slow-wave sleep (SWS) and REM sleep.

Sleep patterns were also studied during the two seasons in African sportsmen in baseline sedentary condition and after exercise with and without rehydration (36). All conditions were randomly assigned. The exercise programme consisted of 3 sequences of cycling, beginning with 10 min at 30 % of MAP, followed by an exhaustive supramaximal effort (1 to 2 min at 130 % of MAP), and ending with a 10 min recovery at 30 % of MAP. Polysomnography was recorded for 2 consecutive nights in each condition. The first session however, regardless of the exercise condition, included one additional familiarization night.

Baseline slow-wave sleep and REM sleep were high (Fig. 6). In the hot season, there was an overall increase in slow-wave sleep, due primarily to an increase in stage 4. Conversely, stage 2 decreased. This effect was also observed after exercise with rehydration, but was absent in the non-hydration condition. This distinction between the rehydrated and non-rehydrated condition is thought to be due to the greater stress which may accompany an exercise-induced relative dehydration. This would also lead to a relative increase in body temperature, as drinking while exercising limits this increase (33). The effects observed were diachronic in baseline and exercise with rehydration conditions. They may have been synchronic in the heat after exercise without rehydration.

Compared to baseline values, REM sleep (Fig. 6) was lower in the cool season after exercise in both hydration conditions due to shorter phases. This decrease in REM sleep could also be_related to a stress reaction occurring or persisting during the night (9).However, it did not occur in the hot season. This difference may be related to the interaction of exercise and heat acclimatization on water balance regulation. Born et al. (3) demonstrated recently that REM sleep is reduced by vasopressin. Both heat exposure and exercise are known to increase plasma vasopressin (34) whereas vasopressin secretion is lowered in heat acclimated subjects (15).

These data emphasise the complexity of the sleep-wake modifications after exercise, with interactions between hyperthermia, water balance and stress.

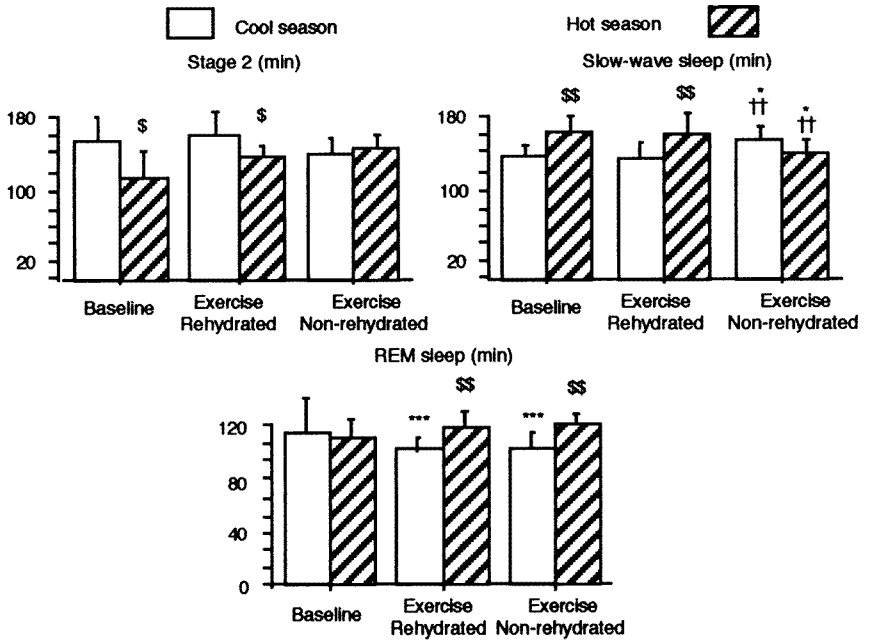

Fig. 6. Combined effect of exercise with and without rehydration and seasonal heat variations on slow-wave sleep and REM sleep in African sportsmen. Significant variations between seasons ($: p<0.05 $$: p<0.02, $$$: p<0.01), between exercise and baseline data (*: p<0.05) and between the two hydration conditions (†: P<0.05; ††: p<0.02; †††: p<0.01) are indicated.

SLEEP IN POLAR CLIMATES

In Antarctica, polysomnograms were recorded in 8 men wintering on the French base of Dumont d'Urville, which is situated under the antarctic circle (10). The base enjoys a microclimate and has been called the "Côte d'Azur" of the Antarctic and was our "island in the sun". As soon as the sea ice permitted, the winterers performed daily outings to visit the magnificent glacier of the Astrolabe, or our neighbours, the Emperor pinguins in winter, and the seals in the spring. This led to a progressive increase in physical fitness accompanied by a progressive increase in slow-wave sleep. These results differ from those of other antarctic polysomnographic studies. At the south pole (27, 46), the base is situated at an altitude of 2,804 m, ambient temperature averages -51 °C and the polar night lasts for 6 months. Slow-wave sleep is highly disturbed and even disappears during the winter. REM sleep is also decreased due to altitude periodic breathing. At Halley Bay (39), situated on an coastal iceshelf, far south of the antarctic circle, slow-wave sleep decreases during the wintering months with the polar night. At Mirnyy (2), under the arctic circle, the Russians reported no change in sleep patterns in winterers living on their coastal base. We attribute the differences with our study to the exceptional attractiveness of Dumont d'Urville's surroundings, which lead to an improvement of physical fitness due to daily outings.

Polysomnography and body temperatures were also recorded in the Arctic, where subjects slept under unheated tents for 10 to 16 consecutive nights (5, 6, 7). Compared to thermoneutral conditions, sleeping in the cold occasioned a large decrease in rectal temperature, which reached 34.9 °C in the middle of the night. Slow-wave sleep was preserved, as it occurred during the first half of the night. However, when the subject was in hypothermia, he could not maintain sleep anymore. Many awakenings interrupted lighter sleep made of stage 1 and stage 2. REM sleep episodes occurred at the same time as they did in the thermoneutral condition. However, REM sleep could not be maintained. This may be related to the fact that body movements and shivering are suppressed during REM sleep.

REM sleep deprivation, expressed as a percent of baseline values, was proportional to the intensity of cold exposure and also proportional to the excretion of 17-OHCS during the night. Therefore, REM sleep shortening was synchronic of the stress reaction.

Three of our subjects had been preacclimated to cold by 9 cold bath sessions of one hour in water at 10 °C. Contrary to our subjects, they did not show any increase in nocturnal diuresis, in 17-OHCS excretion or in noradrenalin excretion. They also did not have any change in their sleep patterns, especially no REM sleep deprivation. This demonstrated that acclimation can prevent the synchronic stress reaction to occur.

SLEEP AS A STRESS REACTION

Jouvet and his group (14, 29) proposed recently an explanation of the intervention of the nervous system in stress reactions following sleep deprivation. In the rat and in the cat immobilization induces sleep deprivation with a stress followed by a rebound in REM sleep and slow-wave sleep. When sleep deprivation is obtained by cuddling the animal, there is no stress and no rebound phenomenon. Stressful sleep deprivation induces an increase of axonal release of serotoninergic neurons ending in the arcuate nucleus (26), one of the main hypothalamic structures which produce proopiomelanocortin. After successive cleavages, this large protein gives ACTH and its two derivatives, a-MSH (melanostimulin) and CLIP (cotic tropin-like intermediate lobe peptide) which represent somnogenic peptides. Such peptides are in turn released in the raphe nuclei where they induce a dendritic release of serotonin and an autoinhibition of serotoninergic neurons, leading to a rebound in REM sleep (14).

Therefore (Fig. 7), when animals are sleep deprived using a stressful technique, a central response involving the arcuate nucleus induces a diachronic REM sleep rebound. When sleep deprivation is gentle, there is no such "central" response and no rebound phenomenon occurs. This is also observed after a lesion of the arcuate nucleus in hypophysectomised rats (50). Such a mechanism could be involved in the changes in sleep patterns in man, not only after sleep deprivation but also after exercicse or exposure to extreme environments.

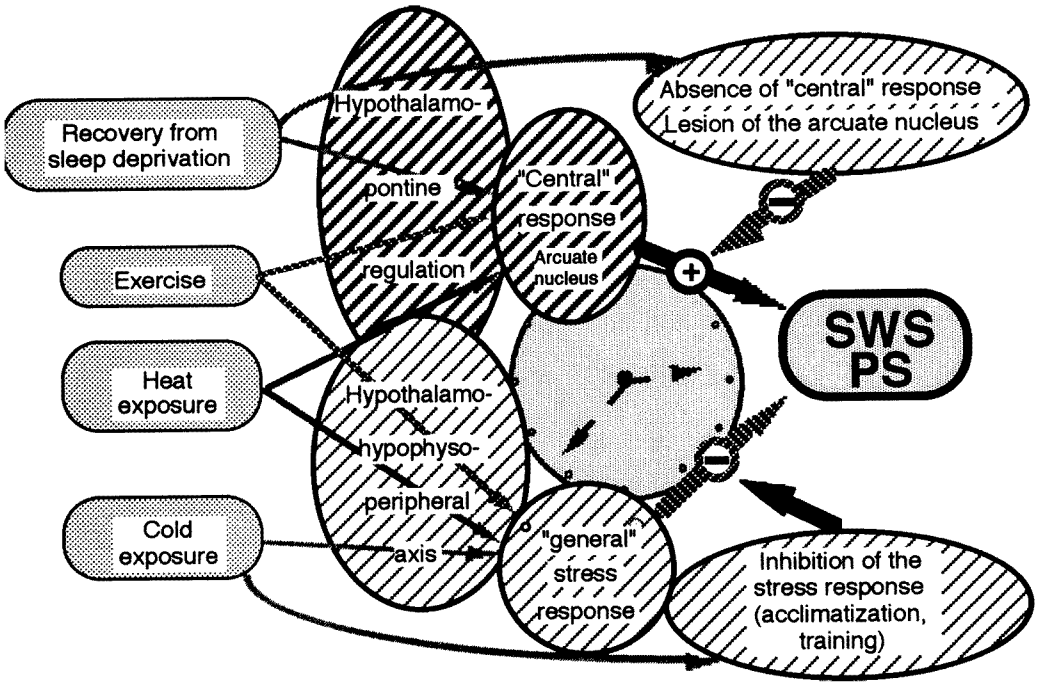

Fig. 7. Recapitulative interpretation of the stress-induced sleep changes after sleep deprivation, exercise and exposure to extreme environments (see text for legend).

Exercise has two effects. The diachronic enhancement of slow-wave sleep is observed in the absence of activation of the adrenal cortical glands and could therefore use the central POMC-serotonin pathway. This effect is observed in moderately trained subjects and in athletes performing a tolerable exercise. A diachronic (such as a decrease in total sleep time and in slow-wave sleep) and synchronic (such as a decrease in REM sleep) sleep disruption would occur when this pathway is overloaded or bypassed, leading to the activation of

the hypothalamo-pituitary-adrenal axis. This is observed when the exercise load is too important for the subject or when the subject is not used to the exercise conditions.

A similar regulation is observed after daytime heat exposure. The diachronic increase in slow-wave sleep would be due to the activation of the central pathway. The synchronic decrease in slow-wave sleep is observed when the subject sleeps in an unusually hot environment.

Cold exposure at night leads to synchronic stress reactions with the activation of stress hormones. The synchronic stress reaction can be limited by previous acclimation or training.

In conclusion, when the brain can deal with the stressful situation, the diachronic increase in slow-wave sleep and/or REM sleep occurs. When this pathway is overloaded, the classical stress reaction occurs with diachronic and synchronic disruptions of sleep architecture. In any case, we believe that the enhanced slow-wave sleep after exercise in temperate climates and in people living in a tropical country, both in sedentary conditions or after exercise, is beneficial. It may serve to lower energy expenditure, as oxygen consumption is at its lowest during slow-wave sleep (17, 46), but also to lower body heat content. This is achieved by an increase in evaporatory heat loss concomitant with slow-wave sleep (17, 18, 37) and by a lowering of core temperature, as was demonstrated in a patient with an aplasia of sweat glands (11). The lowering in metabolic brain activity during slow-wave sleep would allow the occurrence of REM sleep, a state of high energy consumption (29). Furthermore, REM sleep occurs preferentially when core temperature is low (30). In man, it is a state of poor thermal regulation, with an inhibition of sweating in the heat (18) and shivering in the cold (6), leading to increases in skin and core temperatures (18). Therefore, body core cooling during slow-wave sleep may be essential in permitting REM sleep to occur after external or internal heat load.

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